CHAPTER

2

ANATOMY AND PHYSIOLOGY OF ADULT FRICTION RIDGE SKIN

Alice V. Maceo

CONTENTS

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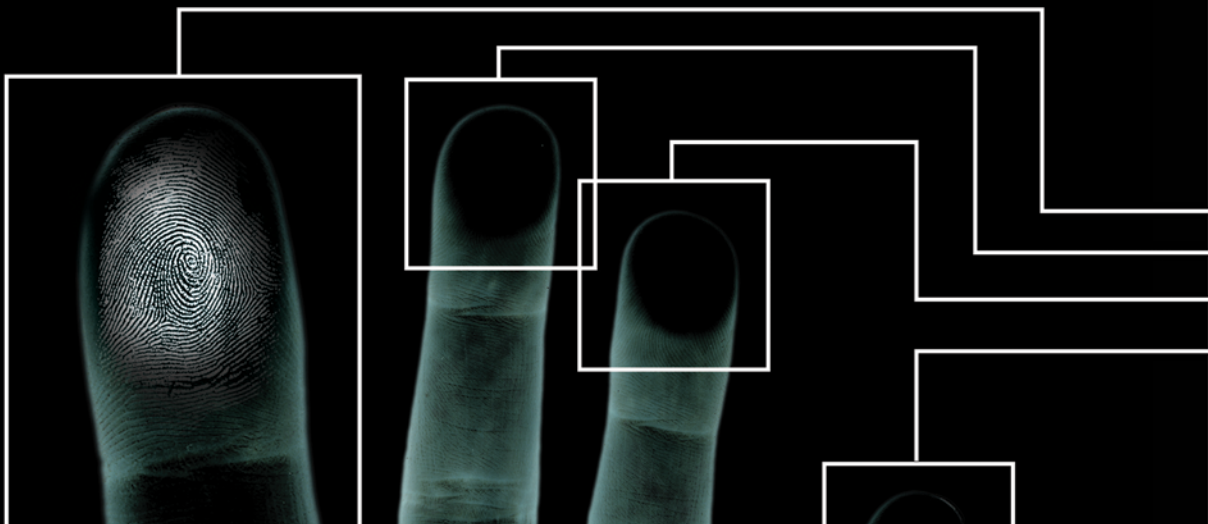
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2.1 Introduction

The anatomy and physiology of the friction ridge skin form the basis for several critical elements that underlie the examination process. The anatomy and physiology explain how the features of the skin persist, how the features of the skin age, how the skin responds to injury, and why scars that form are unique. Another element explained by the structure of the skin is the mechanics of touch. Understanding how the friction ridge skin reacts when it contacts a surface can provide valuable assistance during the examination of friction ridge impressions.

2.2 Anatomy

2.2.1 Outer Morphology of Friction Ridge Skin

The outer morphology of the friction ridge skin is a direct reflection of its function. The ridges and sweat pores allow the hands and feet to grasp surfaces firmly, and the creases allow the skin to flex. Ridges, creases, and mature scars of the friction ridge skin are durable morphological features.

Warts, wrinkles, blisters, cuts, and calluses may also appear on the friction ridge skin and are frequently transient morphological features. The anatomy and physiology of a feature determine whether the feature is durable or transient in nature. Figure 2–1 is an image of a left palm displaying the normal morphology of friction ridge skin.

2.2.2 General Anatomy of Skin

The skin is an organ composed of three anatomical layers: epidermis, dermis, and hypodermis. These anatomical layers together function to provide the body with a protective barrier, body temperature regulation, sensation, excretion,

FIGURE 2–1

*Friction ridge skin
of the left palm.*

immunity, a blood reservoir, and synthesis of vitamin D (Tortora and Grabowski, 1993, p 127).

The outer layer of skin is the epidermis. The epidermis prevents water loss through evaporation, acts as a receptor organ, and provides a protective barrier for the underlying tissues. Melanocytes, the pigment-producing cells of the epidermis, play a key role in the protective barrier. The pigmentation produced by the melanocytes shields the DNA of the keratinocytes (primary cell type of the epidermis) from the sun's harmful rays. Additionally, the melanocytes are responsible for the synthesis of vitamin D (Freinkel and Woodley, 2001, p 120).

The dermis is a layer of connective tissue that supports the epidermis. It is a network of cells, fibers, blood vessels, and gelatinous material that provides structural support and nourishment for the epidermis. The dermis serves as a blood reserve and participates in sensory reception and temperature regulation.

The hypodermis lies under the dermis and is a loose connective tissue that contains a pad of adipose cells (fat) that contour the body and serve as an energy reserve. Fibers link the epidermis to the dermis and the dermis to the hypodermis.

The only skin appendage of the friction ridge skin is the eccrine sweat gland. Although sweat glands are distributed over almost the entire skin surface, the friction ridge skin has the highest concentration of eccrine glands, 2500–3000/2.5 cm^2 (Freinkel and Woodley, 2001, p 49). The sweat glands of the friction ridge skin are also the largest on the body. Eccrine sweat glands participate in temperature regulation by secreting sweat and assist in the

excretion of metabolic waste (e.g., urea) (Junqueira and Carneiro, 2003, p 369).
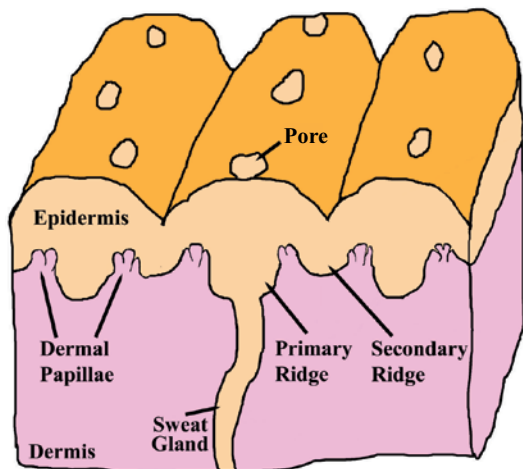
2.2.3 Structure of Friction Ridge Skin

The ridges and furrows on the surface of the friction ridge skin are firmly rooted in the dermis by primary ridges (under-the-surface ridges) and secondary ridges (under the valleys). Figure 2–2 illustrates the structure of friction ridge skin. The primary and secondary ridges are interlocked with the dermis to provide support and strength to the friction ridge skin. Additionally, sweat glands extend from the primary ridges and are anchored in the dermis or hypodermis.

2.2.4 Epidermis

The epidermis is described as a "stratified, continually renewing epithelium that exhibits progressive differentiation (keratinization, cornification) in a basal to superficial direction" (Freinkel and Woodley, 2001, p 19). In other words, the epidermis is a layered tissue that must constantly replace the cells leaving the surface. New cells are generated in the basal layer and pushed toward the surface. As the cells move toward the surface, they undergo sequential changes in chemical composition.

The epidermis is composed of several different types of cells: keratinocytes, melanocytes, Langerhans cells, and Merkel cells. The keratinocytes are the cells that undergo differentiation and are lost at the surface. The epidermis is the protective barrier; it is imperative that the skin balance the number of new keratinocytes created with the number of keratinocytes leaving the surface. This balance is achieved by communication and adhesion.

FIGURE 2–2

Structure of friction ridge skin.
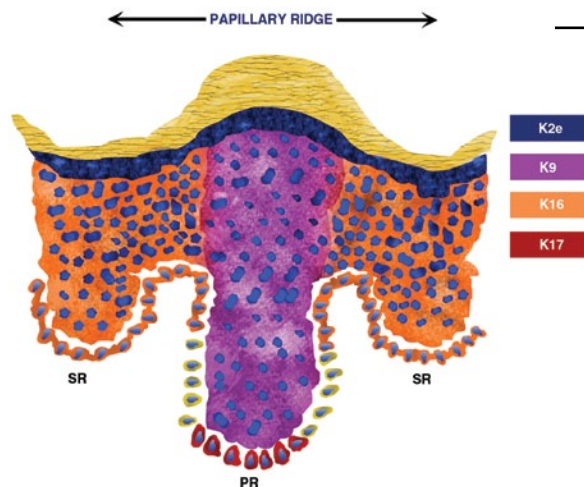
FIGURE 2–3

Distribution of keratin in the primary (PR) and secondary (SR) ridges. Keratin K9 is predominantly expressed in the suprabasal layer and stratum spinosum of the primary ridges. Keratin K17 is expressed in clusters in the basal layer of the primary ridges. Keratin K16 is expressed in the secondary ridges. (Artwork by Brandon Smithson, re-drawn from Swensson et al. (1998), p 773.)

2.2.5 Keratinocytes

The primary cell of the epidermis is the keratinocyte. Keratinocytes account for 90–95% of the epidermal cells (Freinkel and Woodley, 2001, p 19). Even though keratinocytes change in chemical composition as they reach the surface, all keratinocytes are distinguishable by the presence of keratin intermediate filaments.

Keratin is a durable protein organized into bundles (filaments) that extend throughout the cell and provide structural support. Keratin reinforces the skin cells so that they do not break when subjected to physical stress. There are about 20 varieties of keratin distributed throughout the epidermis, designated K1 through K20 (Freinkel and Woodley, 2001, p 20). The keratinocytes of the friction ridge skin express keratins not expressed elsewhere on the body, specifically K9, K6, and K16 (Swennson et al., 1998, p 770).

Keratinocytes of the friction ridge skin also express a more complex pattern of keratin distribution than the rest of the skin. K9 is found only in the keratinocytes above the basal layer of the primary ridges (Swennson et al., 1998, p 770). The basal keratinocytes in the deepest part of the primary ridges express K17 (Swennson et al., 1998, p 771). The basal keratinocytes along the vertical segments of the primary ridges express K6 (Swennson et al., 1998, p 770). K16 is found only in the keratinocytes of the secondary ridges and in the keratinocytes above the dermal papillae (Swennson et al., 1998, p 771). Figure 2–3 illustrates the keratin distribution in the friction ridge skin.

The differences in the keratin produced and distributed across the friction ridge skin are attributed to the greater amount of mechanical stress on the friction ridge skin (Swennson et al., 1998, p 767). The keratin produced in the

FIGURE 2–4

Layers of the epidermis.
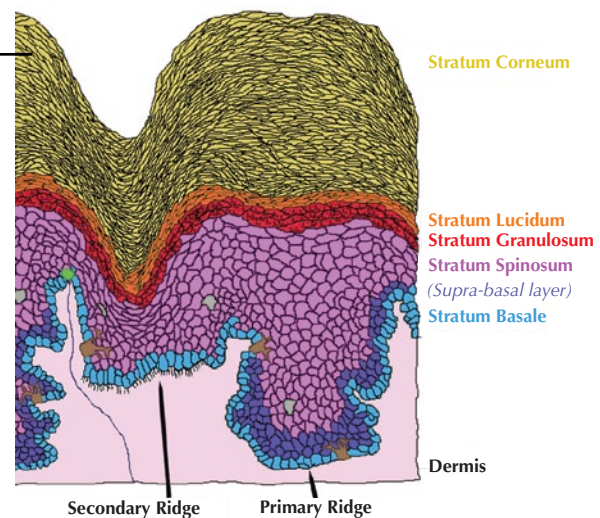
cells of the primary ridges (K9) is more durable than the keratin produced in the secondary ridges (K16). From a mechanical standpoint, the surface ridges withstand most of the compression when the friction ridge skin touches a surface, thereby necessitating enhanced durability. The more pliable keratin produced in the secondary ridges allows the furrows to act as a hinge between the stiffer surface ridges (Swennson et al., 1998, p 772).

2.2.6 Layers of the Epidermis

Figure 2–4 is a color-coded illustration of the five layers of keratinocytes in the friction ridge skin epidermis: stratum basale, stratum spinosum, stratum granulosum, stratum lucidum, and stratum corneum. There is an informal layer, the suprabasal layer, between the stratum basale and the stratum spinosum in the primary ridges.

Nearly all the cells illustrated in Figure 2–4 are keratinocytes. The only exceptions are the occasional brown, grey, and green cells that represent the melanocytes, Langerhans cells, and Merkel cells, respectively.

The layers of the epidermis are named on the basis of microscopic appearance of the keratinocytes in slide preparations. The keratinocytes change in appearance and composition as they are pushed toward the surface and undergo differentiation. During the stages of differentiation, the cells become keratinized (filled with keratin).

2.2.6.1 Stratum Basale. The stratum basale is the innermost layer of the epidermis and consists of a single layer of keratinocytes with occasional melanocytes and Merkel cells.

The keratinocytes in the basal layer continually divide and are the wellspring of all the keratinocytes in the upper layers.

Figure 2–5 is an image of two adjacent basal keratinocytes. Each keratinocyte contains a large nucleus. The nucleus consists of a lighter-stained chromatin and a darker-stained nucleolus. Chromatin is the active DNA specific for that particular cell type (keratinocyte in this instance). The nucleolus is compacted DNA responsible for synthesizing ribosomes. Ribosomes are structures in the cell that help build proteins. The basal cells are connected to the basement membrane zone by hemidesmosomes. The hemidesmosomes link the basal cells to the dermis via the basal lamina. The basal lamina is broken down into two regions: lamina lucida and lamina densa. Desmosomes and focal tight junctions attach the basal keratinocytes to each other. There are small spaces between the cells. These intercellular spaces allow nutrients and signals that have passed from the dermis via the basement membrane zone to diffuse throughout the keratinocytes of the basal layer.

Basal Cell Mitosis. When a basal keratinocyte divides, it undergoes mitosis. Mitosis is the mechanism by which a cell replicates its DNA, the two copies of the DNA migrate to different sides of the cell, and the cell physically separates into two. Each cell contains a complete copy of the DNA. When a basal keratinocyte divides in the epidermis, the original cell remains in the basal layer (cell A in Figure 2–6) and the newly generated cell sits on top of it (cell B in Figure 2–6). When the basal keratinocytes divide again, the first generated cell (B) is displaced into the stratum spinosum by the newly generated cell (cell C in Figure
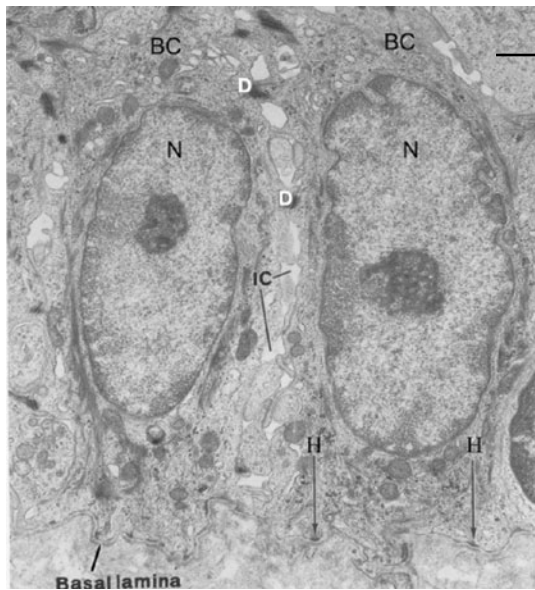
FIGURE 2–5

Two adjacent basal cells (BC), each containing a large nucleus (N). The basal lamina (lamina lucida and lamina densa) lies just below the plasma membrane of the basal keratinocytes. Hemidesmosomes (H) occur regularly along the plasma membrane. Intercellular spaces (IC) are spaces between cells where the cells are not attached by desmosomes (D). Magnification = 2680 X. (Reprinted with permission from Montagna and Parakkal (1974), p 28.)
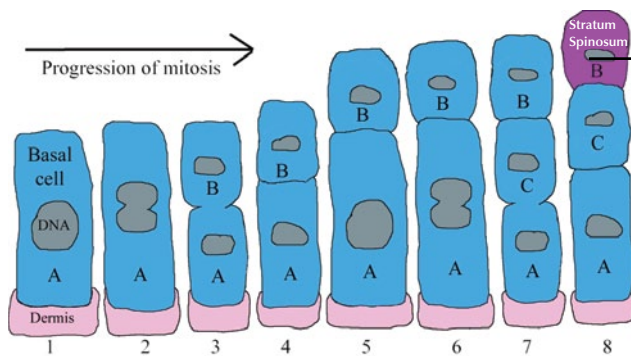
FIGURE 2–6

Sequence of mitosis of basal keratinocytes: (1) cell A replicates its DNA; (2) the DNA is pulled to opposing ends of the cell; (3) cell A divides; (4) cell B is created; (5) cell A replicates its DNA again; (6) the DNA is pulled to opposing ends of cell A; (7) cell A divides to create cell C; (8) cell C pushes previously generated cell B upward, where it begins to differentiate and becomes part of the stratum spinosum.

2–6). The cycle continues, each new cell pushing the older cells toward the surface of the epidermis.

Basement Membrane Zone. The keratinocytes of the stratum basale are associated with the dermis via the basement membrane zone. The basement membrane zone contains elements of both the epidermis and dermis. In addition to providing structural support to the skin, the basement membrane zone is the filter through which nutrients pass from the dermal blood vessels to the basal keratinocytes (Freinkel and Woodley, 2001, p 133).

The basement membrane zone includes the portion of the plasma membrane of the basal keratinocytes that sits on the dermal–epidermal junction. As shown in Figure 2–7, the basal keratinocytes have specialized attachment plaques, termed hemidesmosomes, that project anchoring filaments down toward the dermis (Freinkel and Woodley, 2001, p 134). The area just below the basal cells containing these anchoring filaments is called the lamina lucida.

The dermis contributes the lamina densa and sublamina densa fibrillar zone to the basement membrane zone. The lamina densa contains protein (e.g., collagen fibers). The filaments of the hemidesmosomes in the lamina lucida are interwoven with the fibers of the lamina densa (Freinkel and Woodley, 2001, p 136). The sublamina densa fibrillar zone is the uppermost portion of the dermis and contains elastic fibers, additional collagen fibers, and anchoring plaques (Freinkel and Woodley, 2001, p 145). The fibers and anchoring plaques of the sublamina densa fibrillar zone are interwoven with the fibers of the lamina densa.

The hemidesmosomes of the basal keratinocytes and the interlocking fibers throughout the basement membrane zone prevent the basal cells from migrating. The basal keratinocytes are locked down to their position in the epidermis.

Anchoring Cell Junctions: Desmosomes and Focal Tight Junctions. The keratinocytes of the basal layer, and throughout the layers of the epidermis, are tightly

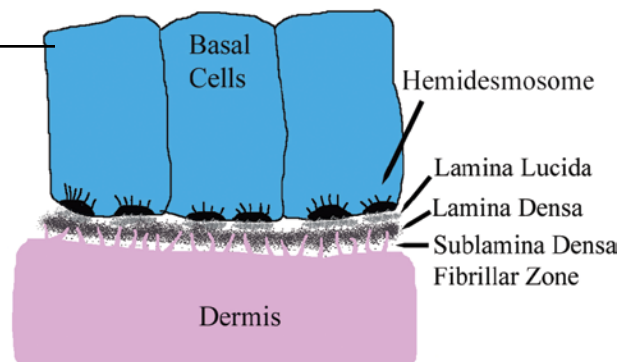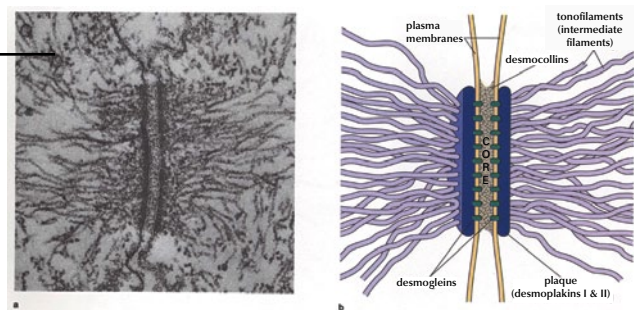**FIGURE 2–7**

Basement membrane zone.

FIGURE 2–8

SEM and schematic of a desmosome linking adjacent skin cells of a salamander. Magnification = 5500 X. (Reprinted with permission from Wolfe (1993), p 257.)


bound to one another via desmosomes (Junqueira and Carneiro, 2003, p 370) and focal tight junctions (Tortora and Grabowski, 1993, p 97). Desmosomes are round plaques that bind together the plasma membranes of adjacent cells. Figure 2–8 shows (a) a scanning electron microscope (SEM) image and (b) a schematic of a desmosome. Keratin fibers extend from the desmosome plaque to the interior of each cell, creating an interior scaffold that supports the cell (Wan et al., 2003, p 378).

Desmosomes exist between cells throughout the entire epidermis (friction ridge skin and nonfriction ridge skin). There is, however, variation. Desmosomes vary in size, depending on the body location of the skin. The desmosomes between the keratinocytes of the friction ridge skin are larger than those of nonfriction ridge skin (Wan et al., 2003, p 384). Along with larger desmosomes, the keratinocytes of the friction ridge skin also have a greater density of keratin (Wan et al., 2003, p 379). The increase in the size of the desmosomes and density of keratin indicates that desmosomes are site specific, depending on the amount of physical stress the particular area of skin must endure (Wan et al., 2003, p 386).

Desmosomes also show variation within the layers of the epidermis. Desmosomes undergo modifications as the cells progress outward from the basal layer of the epidermis. In the friction ridge skin, the desmosomes increase in size as the cells enter the stratum spinosum (Wan et al., 2003, p 385). Desmosomes are continually reinforced as the cells are pushed toward the surface. Upon reaching the outer portion of the stratum corneum, the desmosomes are broken down to release the cells from the surface (Freinkel and Woodley, 2001, p 25).

Focal tight junctions (Figure 2–9) are small "spot welds" of the cells' surfaces (Flaxman and Nelson, 1974, p 329). The cell membranes of adjacent cells are fused together, eliminating intercellular space. Focal tight junctions provide additional anchoring between cells and provide a low-resistance electrical pathway for communication between cells (Cavoto and Flaxman, 1972, p 373).

Basal Cell Heterogeneity. The basal keratinocytes of the primary ridges are structurally different from the basal cells of the secondary ridges. The basal keratinocytes of the primary ridges contain less keratin than the basal cells of the secondary ridges. The junction of the basal cells of the
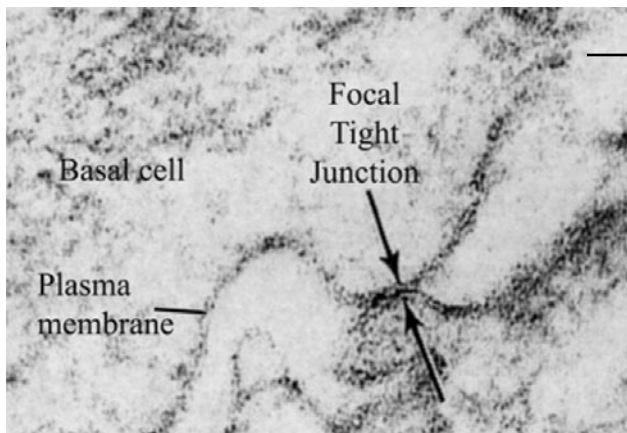
FIGURE 2–9

Electron micrograph of a focal tight junction between adjacent keratinocytes.

Magnification = 12500 X. (Reprinted with permission from Cavoto and Flaxman (1972), p 372.)
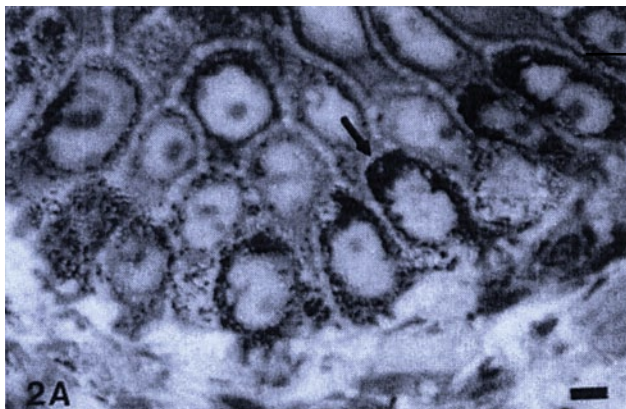
FIGURE 2–10

Basal cells of the primary ridges. Scale bar is 10 µm. (Reprinted with permission from Lavker and Sun (1983), p 123.)

primary ridges with the basement membrane is slightly un-dulated (Figure 2–10), whereas basal cells of the secondary ridges contain long projections that extend deep into the dermis (Figure 2–11) (Lavker and Sun, 1982, p 1240).

The differences in the structure of the basal cells in the primary and secondary ridges explain their differences in function. The basal cells of secondary ridges, with long projections into the dermis, serve an anchoring function (Lavker and Sun, 1982, p 1239). The basal cells of the primary ridges have a morphology similar to stem cells and can be induced to multiply by tissue demand or injury (Lavker and Sun, 1982, p 1239). The basal cells also differ in the rate at which they multiply. The basal cells of the secondary ridges divide more frequently than the primary ridges because the basal cells of the primary ridges give rise to cells that divide in the suprabasal layer.

Suprabasal Layer. The basal keratinocytes of the second-ary ridges continuously divide—each basal cell dividing to push one cell at a time into the stratum spinosum. The basal cells of the primary ridges behave a little differently.

The basal keratinocyte of the primary ridge divides to cre-ate a new cell. This new cell does not immediately enter the stratum spinosum and commit to differentiation. The newly generated cell, termed a transient amplifying cell, undergoes a couple of cell divisions while it sits in the suprabasal layer (Lavker and Sun, 1983, p 121). After cell divisions are complete, the transient amplifying cells are pushed upward into the stratum spinosum and begin dif-ferentiation. More cells are produced in the primary ridges than in the secondary ridges because of the transient amplifying cells. The cells of the primary ridges maintain the surface ridges, where more cells are needed because of greater abrasion.

2.2.6.2 Stratum Spinosum. As the keratinocytes are pushed toward the surface, they begin to undergo differen-tiation. The cells become polyhedral in shape and desmo-somes (cell junctions) are reinforced. Keratin production is increased, and the keratin filaments are organized concentrically around the nucleus and extend into the desmosomes (Freinkel and Woodley, 2001, p 23). New

FIGURE 2–11

Basal cells of the secondary ridges. Scale bar is 10 µm. (Reprinted with permission from Lavker and Sun (1983), p 123.)
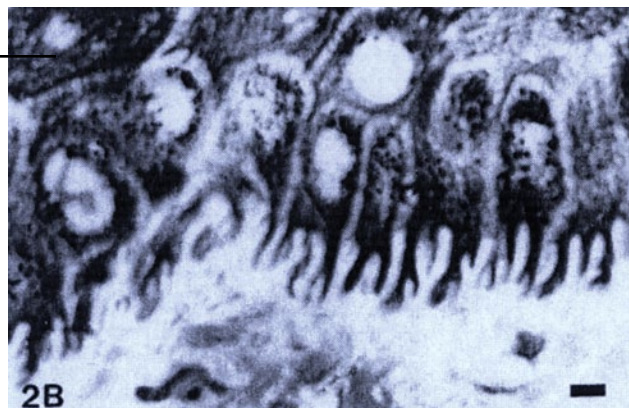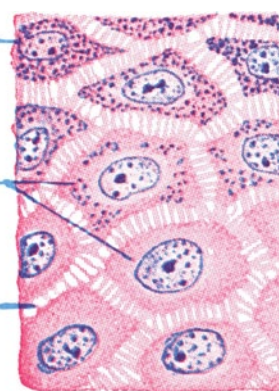
FIGURE 2–12

Cells of the stratum spinosum and stratum granulosum (with keratohyalin granules). Magnification = 1400 X. (Reprinted with permission from Eroschenko (1993), p 127.)

structures, lamellar granules, appear in the cells as the cells are pushed toward the limit of the stratum spinosum. Lamellar granules are pockets of lipids that first appear in the stratum spinosum but do not become active until the cells reach the stratum granulosum (Freinkel and Woodley, 2001, p 24). Figure 2–12 is a microscope slide preparation of the keratinocytes of the stratum spinosum and stratum granulosum. The stratum spinosum is so named because of the spiny appearance of the cells in microscope slide preparations. During the process of making the slide, the cells dehydrate, causing them to shrink away from one another. The spines are where the desmosomes are still holding the cells together.

2.2.6.3 Stratum Granulosum. As the cells are pushed toward the surface, they continue structural and chemical modification. Keratinocytes entering the stratum granulosum contain characteristic keratohyalin granules (Figure 2-12). The keratinocytes are programmed to fill with keratin; the keratohyalin granules contain proteins (profilaggrin, keratin, and loricrin) that facilitate the process (Freinkel and Woodley, 2001, p 23). The lamellar granules become active as the cells reach the upper portion of the stratum granulosum. The lamellar granules release their lipid content

into the space between the cells. The lipids coat the cells, providing the skin with a hydrophobic barrier (Freinkel and Woodley, 2001, p 24).

2.2.6.4 Stratum Lucidum. The keratinocytes undergo an abrupt transition to the stratum lucidum. The cells are keratinized and have completed their programmed cell death (Freinkel and Woodley, 2001, p 24). Although the cells are no longer living, chemical activity continues inside the cells as the final modifications are made to the keratin.

2.2.6.5 Stratum Corneum. With layer upon layer of non-viable, terminally differentiated keratinocytes, the stratum corneum is the significant epidermal layer that allows skin to act as a major barrier. The arrangement of keratinocytes is described as a "brick-and-mortar model". The keratin-filled cells (bricks) are surrounded by the lipids (mortar) secreted while the cells were in the stratum granulosum (Freinkel and Woodley, 2001, p 25). Although they are dead, the cells of the stratum corneum continue to undergo modification as they are pushed from the deeper portion of the stratum corneum to the surface of the skin. The cells in the deeper portion of the stratum corneum are thicker and have more densely packed keratin, a weaker cell

FIGURE 2–13

Surface of the friction ridges showing the cells shedding from the surface. (Reprinted with permission from Montagna and Parakkal (1974), p 25.)

membrane, and more cell-to-cell attachments (Freinkel and Woodley, 2001, p 25). As the cells are pushed toward the surface, the cell membrane becomes more rigid and the desmosomes are degraded. These changes allow the cells to shed when they reach the surface (Figure 2–13).

2.2.7 Nonkeratinocytes

Communication of the keratinocytes with the melanocytes, Langerhans cells, and Merkel cells is necessary for the skin to function properly.

Melanocytes produce the pigments that are deposited into the keratinocytes. This pigment, melanin, protects the genetic material of the keratinocytes from ultraviolet damage (Junqueira and Carneiro, 2003, p 374). Melanocytes reside in the basal layer of the epidermis and, in addition to providing the surrounding keratinocytes with melanin, produce vitamin D.

The Langerhans cells are an extension of the body's immune system. Upon exposure to invading bacteria, Langerhans cells initiate an alert that causes the body to recruit more aggressive immune cells (T cells) to attack the invaders (Freinkel and Woodley, 2001, p 30).

The Merkel cells are an extension of the nervous system and participate in the transmission of the sensation of touch: "shape, size, and texture of objects and two-point discrimination" (Dillion et al., 2001, p 577). Merkel cells occur sporadically in the basal layer of the epidermis and are associated with free nerve endings from the dermis.

2.2.8 Dermis

2.2.8.1 Papillary Dermis. The dermis is the connective tissue that supports the epidermis and binds it to the

hypodermis. The dermis is composed of two layers: the papillary layer and the reticular layer. The outer papillary layer is a loose connective tissue containing anchoring fibrils and numerous dermal cells. The anchoring fibrils secure the dermis to the epidermis via the basement membrane zone. The papillary layer of the dermis forms the dermal papillae.

2.2.8.2 Dermal Papillae. Dermal papillae are malleable, peglike projections of the papillary dermis between the primary and secondary ridges. The malleable nature of the dermal papillae is important because the epidermal–dermal junction remodels with age and in response to sheering stress on the surface of the skin (Misumi and Akiyoshi, 1984, p 53; Chacko and Vaidya, 1968, p 107). During the remodeling, the epidermis forms sheets of tissue that cross-link adjacent primary and secondary ridges. These sheets of tissue are called anastomoses. As the epidermal anastamoses form, the dermal papillae are molded into increasingly more complex structures (Hale, 1952, p 153). The detail of Figure 2–14 illustrates the dermal papillae and anastomoses. The formation of dermal papillae and epidermal anastomoses increases the surface area of attachment between the epidermis and dermis, thereby increasing the bond between the epidermis and dermis.

2.2.8.3 Reticular Dermis. The reticular dermis is a compact connective tissue containing large bundles of collagen and elastic fibers. The organization of these fibers provides the dermis with strength and resilience (Freinkel and Woodley, 2001, p 38). The reticular dermis is connected to the hypodermis by a network of fibers.

2.2.8.4 Circulatory System of the Dermis. There are two plexuses of arterial blood vessels in the dermis. One plexus lies between the papillary and reticular dermis and the other between the reticular dermis and the hypodermis

FIGURE 2–14

Cross section of friction ridge skin with detail of the epidermis separated from the dermis to display the dermal papillae and complementary epidermal anastomoses.
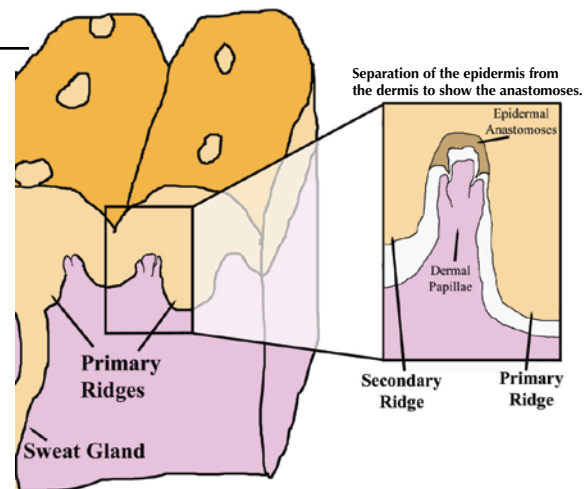

Separation of the epidermis from the dermis to show the anastomoses.

Epidermal Anastomoses

Dermal Papillae

Primary Ridges

Secondary Ridge

Primary Ridge

Sweat Gland

FIGURE 2–15

Circulation system of the skin. (Adapted with permission from Freinkel and Woodley (2001), p 177.)

Dermal papillary loop

Plexus between papillary and reticular dermis

Plexus between reticular dermis and hypodermis
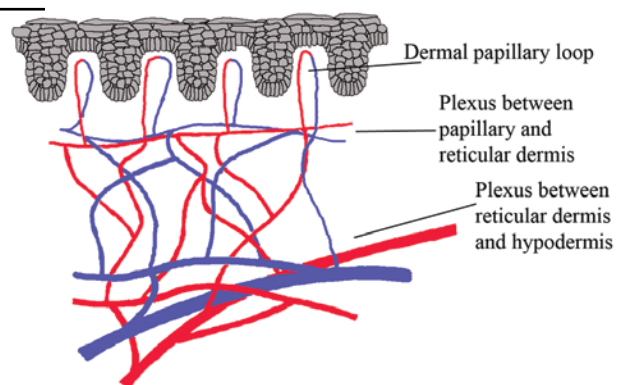
(Junqueira and Carneiro, 2003, p 376). Capillaries extend from the arterial plexus and into the dermal papillae to form the dermal papillary loop (Figure 2–15) (Freinkel and Woodley, 2001, p 38).

Blood passes from the arterial capillaries in the dermal papillae to the venous capillaries. Veins are organized into three plexuses: one associated with each arterial plexus and a third plexus in the middle of the reticular dermis (Junqueira and Carneiro, 2003, p 376).

2.2.8.5 Nervous System of the Dermis. A vast network of sensory and autonomic nerve branches innervates the dermis. The autonomic nerve network is responsible for controlling blood flow and glandular secretions (sweat). The sensory system contains receptors for sensations: touch, temperature, pain, and itch (Freinkel and Woodley, 2001, p 153). The dermis participates in sensory perception via free nerve endings, Meissner corpuscles, Ruffini

corpuscles, and Pacinian corpuscles. Free nerve endings and Meissner corpuscles are found in the dermal papillae. Free nerve endings are found in each dermal papilla and provide a rapid response to stimuli (Freinkel and Woodley, 2001, p 157). Meissner corpuscles (Figure 2-16) are found in about every fourth papilla and function as touch receptors (Freinkel and Woodley, 2001, p 160). Pacinian and Ruffini corpuscles are located throughout the dermis and also function in the transmission of pressure (Freinkel and Woodley, 2001, p 158).

2.2.9 Sweat Glands

Although the skin produces several appendages (e.g., hair, nails, sebaceous glands), the eccrine sweat gland is the only appendage of the friction ridge skin. Eccrine sweat glands are found all over the body surface and function primarily in thermoregulation. The sweat glands do not function individually but rather as groups or simultaneously
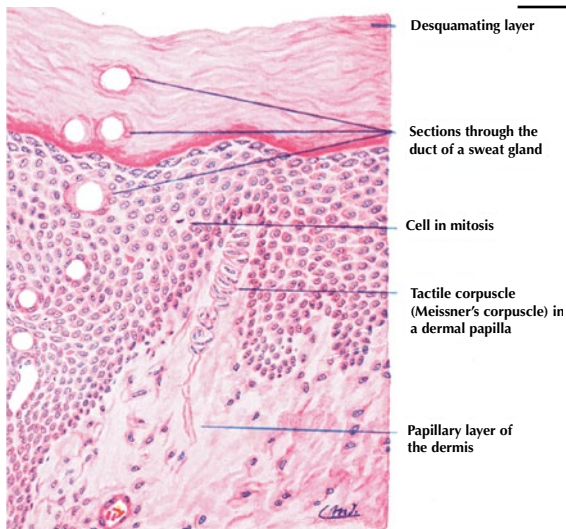
FIGURE 2–16

Section of palm skin showing Meissner's corpuscle in a dermal papilla. Magnification = 100 X. (Reprinted with permission from Eroschenko (1993), p 127.)

Desquamating layer

Sections through the duct of a sweat gland

Cell in mitosis

Tactile corpuscle (Meissner's corpuscle) in a dermal papilla

Papillary layer of the dermis

FIGURE 2–17

Sweat emitting from the pores on the friction ridge skin. (Reprinted with permission from Montagna and Parakkal (1974), p 381.)

over the entire surface of the body (Freinkel and Woodley, 2001, p 47). The sweat glands of the palms and soles are larger, more active, and denser than in any other area of skin. Figure 2–17 is an image of the friction ridge skin sweating.

Eccrine sweat glands are classified as simple tubular glands whose ducts open at the skin surface (Junqueira and Carneiro, 2003, p 380). As shown in Figure 2–18, the coiled secretory portion of the gland is embedded in the dermis or hypodermis, and the duct extends through the epidermis. The fluid secreted by the eccrine sweat glands is predominantly water (99.0–99.5%) (Freinkel and Woodley, 2001, p 71). The remaining constituents of sweat include sodium chloride, potassium, ammonia, urea, lactate, uric acid, creatinine and creatine, amino acids, sugars, immunoglobulin A, epidermal growth factor, and select
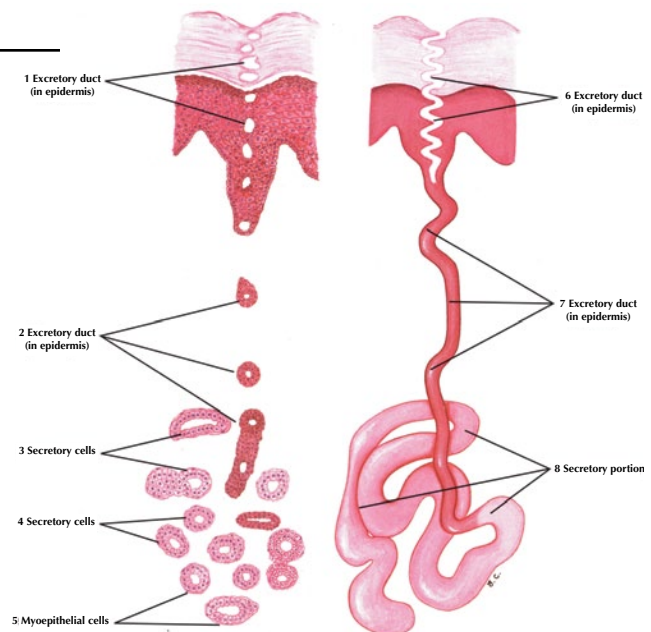
hormones, enzymes, and vitamins (Freinkel and Woodley, 2001, p 71).

2.2.10 Hypodermis

Beneath the fibrous reticular dermis there is an abrupt transition to the adipose tissue of the hypodermis. Adipose (fat) tissue serves as an energy reserve, cushions the skin, contours the body, and allows for mobility of the skin over underlying structures (Freinkel and Woodley, 2001, p 39). The dermis and hypodermis are physically connected through interlocking fibers and share blood vessel and nerve networks (Freinkel and Woodley, 2001, p 39). The primary cell of the hypodermis is the adipocyte. Adipocytes are organized in lobules by fibrous connective tissue and store the subcutaneous fat.

FIGURE 2–18

Sweat gland. (Reprinted with permission from Eroschenko (1993), p 129.)

1 Excretory duct (in epidermis)
6 Excretory duct (in epidermis)
2 Excretory duct (in epidermis)
7 Excretory duct (in epidermis)
3 Secretory cells
4 Secretory cells
8 Secretory portion
5 Myoepithelial cells

2.3 Physiology

The epidermis exists in a dynamic, steady state. Cells lost at the surface must be replaced (dynamic) in order for the skin to maintain (steady) its protective barrier (state). The concept of keeping things the same despite constant input and output of materials and energy is referred to as homeostasis. Homeostasis is defined as "the condition in which the body's internal environment remains relatively constant, within physiological limits" (Tortora and Grabowski, 1993, p 9). Homeostasis is critical to the functioning of all organisms. Homeostasis of the skin is achieved through physical attachments and the careful regulation of cell production in the stratum basale via cell communication.

2.3.1 Physical Attachments

There are structural features of the overall skin and of the skin cells that maintain the structure of the epidermis (even though skin cells are always sloughing at the surface). There are three levels of attachment in the friction ridge skin: the primary/secondary ridge attachment with anastomoses, the basement membrane zone, and cell-to-cell attachments.

2.3.1.1 Primary and Secondary Ridges. The first level of attachment is the topography at the junction of the epidermis and dermis. The alternating system of primary and secondary ridges on the bottom of the epidermis provides general structural support for the surface ridges and furrows. The sweat glands of the primary ridges are firmly attached in the dermis or hypodermis. Additional reinforcement of this system is provided by dermal papillae and epidermal anastomoses.

2.3.1.2 Basement Membrane Zone. The second level of attachment is the basement membrane. The basement membrane is a fibrous sheet that attaches the basal keratinocytes of the epidermis to the underlying dermis. The basement membrane is generated by the basal keratinocytes of the epidermis and the fibroblasts of the dermis. The basal cells of the epidermis have specialized attachment plaques, termed hemidesmosomes, which project fibers down toward the dermis. The dermis projects anchoring fibers back up toward the epidermis. These fibers originating from the epidermal basal cells and from the dermis are interwoven to create the fibrous sheet that locks the epidermis to the dermis. The hemidesmosomes and interlocking fibers prevent the basal cells from migrating. The basal keratinocytes are locked down to their position in the epidermis.

2.3.1.3 Cell-to-Cell Attachments. The third level of attachment consists of the cell-to-cell attachments of the keratinocytes throughout the layers of the epidermis. Desmosomes and focal tight junctions attach the keratinocytes to one another. Desmosomes are reinforced as the cells move from the basal layer to the surface. Upon reaching the outer portion of the stratum corneum, the desmosomes and focal tight junctions are broken down to release the cells from the surface.
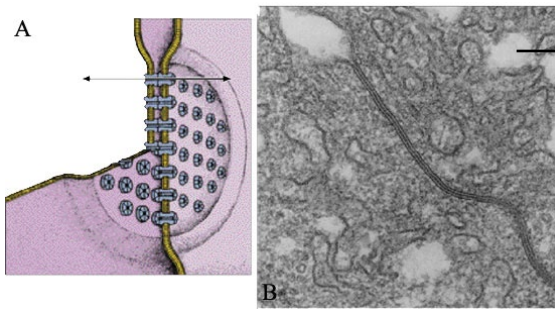
FIGURE 2–19

(A) Model of a gap junction demonstrating the channels that connect the cells; (B) SEM of gap junction between two rat liver cells. Magnification = 59,000 X. (Reprinted with permission from Junqueira and Carneiro (2003), p 74.)

2.3.2 Cell Communication

Skin must maintain the protective barrier while existing in a dynamic steady state (i.e., cells leaving the surface must be replaced). The rate at which basal cells divide in the basal layer must coincide with the rate at which cells are leaving at the surface. There must be a mechanism in place to control the rate of cell division of the basal keratinocytes and to monitor the thickness of the skin. This mechanism is cell communication. The keratinocytes are in constant communication with one another and with the melanocytes, Langerhans cells, and Merkel cells. The keratinocytes are also in communication with the rest of the body via the dermis.

2.3.2.1 Gap Junctions. Rapid communication between cells is achieved via gap junctions. Gap junctions are connections between the cell membranes of adjacent cells that permit the direct exchange of small molecules, ions, and hormones. Figure 2–19 contains a diagram and an electron micrograph of a gap junction between cells. Rapid communication via gap junctions results in the keratinocytes acting in a coordinated manner rather than as independent units (Junqueira and Carneiro, 2003, p 72).

2.3.2.2 Cell Surface Receptors. In addition to the direct cell-to-cell communication through gap junctions, cells also have modified proteins embedded in the outer membrane that can respond to signals sent through the blood or from other cells in the epidermis. When a signal molecule binds to the outer surface of the membrane protein, it causes a cascade of reactions inside the cell to elicit the appropriate response.

2.3.3 Regulation of Keratinocyte Proliferation

2.3.3.1 Cell Cycle. Cell communication is necessary for monitoring and adjusting the rate at which the basal cells divide. The cell cycle describes the stages of DNA replication and cell division. The five phases of the cell cycle are represented as G0, G1, S, G2, and M. G1 is the time gap

that occurs after the cell has divided and before the cell begins replication of its DNA for the next division. G1 is the resting period between mitoses. The duration of G1 is the most variable phase of the cell cycle, and modifications to its duration greatly influence the number of basal cells produced (Freinkel and Woodley, 2001, p 202). During G1, the cell reaches a critical restriction point and monitors conditions to determine whether it will enter the next phase of the cell cycle, the S phase, synthesis. During the S phase, the cell replicates its DNA, a process that takes about 8–12 hours (Freinkel and Woodley, 2001, p 202).

Once replication of the DNA is complete, the cell enters a second gap phase, G2, for approximately 8 hours. During the G2 phase, the cell reaches a second critical restriction point and evaluates the results of DNA replication before entering mitosis (Freinkel and Woodley, 2001, p 203). The M phase, mitotic phase, is the physical division of the cell into two, each containing a complete copy of the DNA.

Upon completion of mitosis, the basal cells may enter into G1 and continue the cell cycle or they may enter G0. Basal cells entering G0 are no longer cycling but may re-enter the cell cycle upon receipt of the appropriate signal (Freinkel and Woodley, 2001, p 203). The new cells created by the basal cells will either withdraw from the cell cycle and begin differentiation or cycle a few more times (transient amplifying cells) before differentiating. The cells that have started to differentiate are the cells entering the stratum spinosum.

2.3.3.2 Regulation of Cell Cycle. There are many opportunities throughout the cell cycle to regulate the rate at which the basal cells undergo mitosis. Signals that stimulate proliferation are received via cell surface receptors. These signals include hormones, proteins, ions (particularly calcium), and vitamins A and D. Once received, the signal triggers the production of two types of partnered proteins inside the cell: cyclins and cyclin-dependent kinases (Freinkel and Woodley,

2001, p 205). The kinases are responsible for advancing the cells through the G1 and G2 phases of the cell cycle. The kinases must bind the appropriate cyclins to accomplish this task. Cyclins are short-lived, unstable proteins. By controlling the availability of cyclins, the ability of the kinases to progress the cells through mitosis is also controlled.

Calcium is also important for a cell's progression through the cell cycle. Calcium binds to a small protein, calmodulin. The calcium–calmodulin complex is a necessary component of the spindle apparatus that separates the two copies of the DNA produced during the S phase of the cell cycle. Calmodulin also makes structural changes inside the cell to induce replication of the DNA during the S phase (Freinkel and Woodley, 2001, p 204).

2.3.3.3 Inhibitors of Mitosis. If the basal cells are responsible for balancing the number of cells produced with the number of cells leaving the surface, there must be some mechanism for them to "know" how many cells are in the outer layers so they can shut down production as needed. This process, common to all living organisms, is called a feedback mechanism.

As the keratinocytes are pushed toward the surface, they undergo radical changes in their internal and external biochemistry. When the cells reach the stratum granulosum, they release the contents of the lamellar granules to provide the "mortar" between the cells. Molecules released by the differentiating cells, referred to as chalones, diffuse through the intercellular spaces and eventually reach the basal cells (Freinkel and Woodley, 2001, p 205). The basal cells, via cell surface receptors, monitor the concentration of chalones. The more cells that differentiate, the higher the concentration of chalones. If the concentration becomes too high, the chalones signal the basal cells to halt the cell cycle. In this manner, the chalones provide feedback to the basal cells regarding the number of differentiating cells in the outer layers.

2.3.3.4 Genetics of Cell Cycle Regulation. Stimulatory signals and inhibitory signals act on oncogenes and tumor suppressor genes, respectively. Oncogenes are the genes that, when translated, generate the proteins necessary for a cell to undergo mitosis. Tumor suppressor genes are genes whose protein products inhibit mitosis. An example of the cell cycle genetic regulation in the epidermis would be as follows: (1) the basal cells bind a stimulatory hormone on a cell surface receptor; (2) a cascade of reactions takes place inside the cell that results in the genes for cyclins

being translated; (3) the production of cyclins activates the kinases, pushing the cells through mitosis; (4) the concentration of chalones rises as the newly generated cells differentiate; (5) chalones diffuse to the basal cells and bind to the appropriate cell surface receptor; (6) a cascade of reactions inside the cells results in the translation of a tumor suppressant gene; and (7) the resultant suppressor protein binds to and inactivates the kinases, thereby halting the cell cycle.

2.4 Persistence of the Friction Ridge Skin

The friction ridge skin persists because of the physical attachments throughout the skin and the regulation of keratinocyte production and differentiation. The three-dimensional morphology of the surface ridge is maintained by the combination of increased cell production in the suprabasal layer of the primary ridges (under-the-surface ridges) and the enhanced anchorage of the basal cells in the secondary ridges (under-the-surface furrows). The basal layer of keratinocytes provides the template for the surface ridges and furrows. Cell communication ensures that basal cell proliferation is stimulated and inhibited in a coordinated manner. As the basal keratinocytes divide, the cell-to-cell attachments ensure that the cells move toward the surface in concert.

2.4.1 Aging of Friction Ridge Skin

Aging is defined by Dr. Barbara Gilchrest as "an irreversible process which begins or accelerates at maturity and which results in an increasing number and/or range of deviations from the ideal state and/or decreasing rate of return to the ideal state" (Gilchrest, 1984, p 5). The friction ridge skin, although durable, undergoes subtle changes as a person ages. The arrangement of the friction ridges does not change; the ridges and furrows maintain their position in the skin. Advancing age has two effects on the friction ridge skin: (1) the surface ridges tend to flatten, making them appear "less sharp" (Okajima, 1979, p 193), and (2) loss of elasticity in the dermis causes the skin to become flaccid and to wrinkle.

2.4.1.1 Flattened Ridges. The friction ridges tend to flatten because of a combination of atrophy of the epidermis and remodeling of the dermal papillae. The remodeling of the dermal papillae is the most striking change in the friction ridge skin. Dermal remodeling continues throughout an
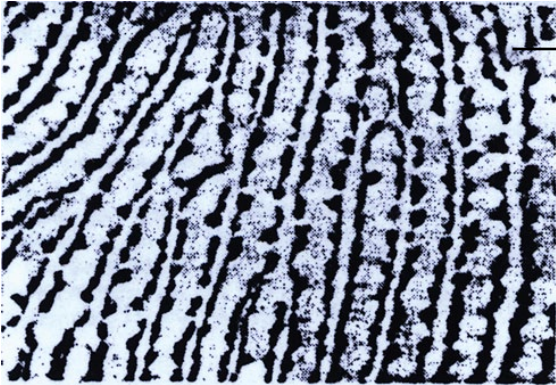
FIGURE 2–20

Dermal surface interdigital area of palm of 30-week-old fetus. (Reprinted with permission from Okajima (1975), p 249.)

FIGURE 2–21

Dermal surface finger apex of an adult. (Reprinted with permission from Okajima (1975), p 249.)

individual's lifetime and varies across the surface of the palm and sole, depending on how much sheering stress has occurred in that particular area. Chacko and Vaidya (1968, p 105) describe three categories of dermal papillae (DRI, DRII, and DRIII) based on the increasing complexity and branching of the papillae. All three types of dermal papillae are found across the palm and sole but show greater variation on the palm (Chacko and Vaidya, 1968, p 107). The greater variation on the palm is attributed to the wider range of uses of the hand compared to the foot.

In Figures 2–20 and 2–21, the epidermis has been removed and the dermal papillae stained with toluidine blue (Okajima, 1975, p 244). The dark-stained areas of Figures 2–20 and 2–21 are the tips of the dermal papillae. Figure 2–20 is the dermal surface of a 30-week-old fetus. Typical of fetal skin, the dermal papillae are arranged in a very orderly double row under each surface ridge.

As the skin ages and is exposed to sheering stress, the existing dermal papillae branch out, and new small papillae form to increase the adhesion of the epidermis to the dermis (Misumi and Akiyoshi, 1984, p 49). Figure 2–21 is the dermal surface of an adult finger. The number of

dermal papillae tends to increase with age, and the papillae become more crowded.

Occasionally, new dermal papillae will also form underneath the furrows of the surface ridges (below the secondary ridges). Dermal papillae that form underneath the surface furrows can range from short and "pebble-like" to the same size as the dermal papillae under the surface ridges (Okajima, 1979, p 193). As the dermal papillae under the furrows become larger, the surface ridges become flatter. Flattening of the surface ridges usually occurs with age (Okajima, 1979, p 193).

The increase in the complexity and number of dermal papillae as a person ages is not reflected in the configuration of the surface ridges and furrows (Misumi and Akiyoshi, 1984, p 53). The epidermis responds to the dermal papillae by forming complementary anastomoses to attach to the branching papillae. The dermal papillae/epidermal anastomoses formation does not affect the basal layer of keratinocytes. That layer is buffered from the dermal changes by the basement membrane and continues to reproduce the surface ridges.

The effects of age on the epidermis also contribute to the flattening of the surface ridges; however, that impact is significantly less compared to the changes in the dermis. The epidermis maintains the thickness of the stratum corneum throughout an individual's lifetime (Lavker et al., 1987, p 46). This is necessary, considering the role of the epidermis as the outer protective barrier. The capacity of the basal keratinocytes to proliferate, however, decreases by 30–50% from the age of 30 to the age of 80 (Gilchrest, 1984, p 21). The slower rate of proliferation results in a thinning of the living layers of the epidermis (stratum basale, stratum spinosum, and stratum granulosum) (Lavker, 1979, p 60).

The remodeling of the dermal papillae, particularly when the dermal papillae form under-the-surface furrows, and the overall thinning of the epidermis contribute to the flattening of the surface ridges that occurs naturally with age. The flattening of the ridges does not affect the sequence and lengths of the surface's ridges and furrows. However, as the ridges flatten, it may be increasingly difficult to follow the ridges and furrows in an impression of the friction ridge skin. Flattening may also diminish the visibility of the edges and contours of the ridges in an impression of the friction ridge skin. It should be noted that the friction ridge skin is quite durable and that the flattening of the ridges occurs slowly, over the course of several decades.

2.4.1.2 Wrinkles. Wrinkles are the result of mechanical changes that take place in the skin as it ages (Kligman et al., 1985, p 41). In other words, there are no special structures formed by the epidermis or dermis at the site of a wrinkle (Kligman et al., 1985, p 40). The overall changes that take place in the skin, particularly in the dermis, as a person ages alter the mechanical properties of the skin. The dermis thins as the network of collagen and elastin fibers becomes compacted. Additionally, the collagen starts to unravel, and the elastin fibers lose their elasticity. The compaction and degradation of the fiber networks in the dermis causes the skin to be "less stretchable, less resilient, more lax, and prone to wrinkling" (Lavker et al., 1989, p 65). The skin becomes loose and simply folds in on itself, creating a wrinkle.

2.4.2 Wound Healing

The friction ridge skin persists throughout an individual's lifetime. The morphology of the friction ridges can be altered only if the basal keratinocyte template is altered. Figures 2–22 through 2–30 are diagrams of a skin model that demonstrate the cellular response of the keratinocytes

to a wound. Figure 2–22 shows the intact skin, and Figure 2–23 illustrates the skin after injury. Upon assault, keratinocytes have been removed and damaged, and the dermis has been injured.

Injury causes the basal keratinocytes to undergo remarkable changes in their structure and physiology to repair the wound. The scar formed by the process of repair results in a new, unique, and persistent feature of the friction ridge skin. The process of wound healing is broken down into three phases, although there is considerable overlap: inflammation, proliferation and tissue formation, and tissue remodeling.

2.4.2.1 Phase I: Inflammation. Inflammation begins immediately after the injury. The disruption of the blood vessels in the dermis causes blood to spill into the surrounding tissue. The platelets from the blood direct the clotting of the blood and send out signals to recruit cells from the immune system and the cells of the dermis (Freinkel and Woodley, 2001, p 282). The immune cells kill bacteria and scavenge damaged cells. The dermal cells (fibroblasts) are concentrated in the wound area to repair the dermis. Additionally, endothelial cells (cells from the blood vessels) begin to repair the damaged blood vessels. It should be noted that the repair of the dermis and epidermis occurs underneath the formed blood clot, although the blood clot is not shown in the following diagrams.

2.4.2.2 Phase II: Proliferation and Tissue Formation. As the fibroblasts and endothelial cells continue to repair the dermis, the basal keratinocytes on the edge of the wound take control of the healing process to start Phase II. As a result of the injury, the basal keratinocytes are suddenly exposed to the dermis by disruption of the basement membrane. Contact with the dermis causes the basal keratinocytes to undergo dramatic changes: The desmosomes and hemidesmosomes dissolve, actin filaments form inside the periphery of the cell, and pseudopodia (footlike projections) are extended from the cell (Rovee and Maibach, 2004, p 61).

The dissolution of the desmosomes and hemidesmosomes releases the basal keratinocytes from their firm attachments. The actin filaments, which act like miniature cell muscles, and the pseudopodia allow the skin cells to crawl across the wound. As the basal keratinocytes at the edge of the wound crawl, the basal keratinocytes behind them divide to create additional cells to help cover the wound (Rovee and Maibach, 2004, p 61).
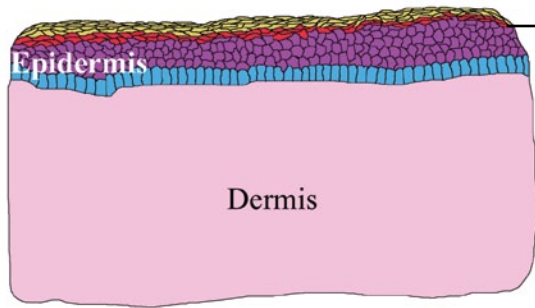
FIGURE 2–22

Intact keratinocyte layers.
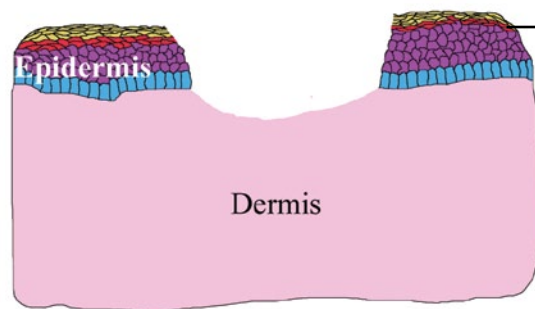
FIGURE 2–23

Injured skin.
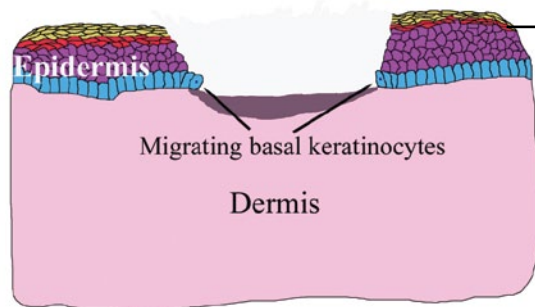
FIGURE 2–24

Repair of the dermis and start of migration of basal keratinocytes at the edge of the wound.

As the opposing sheets of basal keratinocytes move toward one another, the dermis contracts the wound bed to shorten the distance keratinocytes have to migrate to cover the wound (Darby and Hewitson, 2007, p 145). In the friction ridge skin, this contraction creates the classic puckering of the ridges at the scar site. Figure 2–24 demonstrates the repair of the dermis and the beginning of the basal keratinocyte migration. Figure 2–25 demonstrates the puckering of the skin surface as the keratinocytes migrate and the dermis contracts to close the wound. Figure 2–25 also demonstrates the proliferation of the basal keratinocytes behind the migrating cells.

When the leading cells of migrating basal keratinocytes contact each other, they form gap junctions (Flaxman and Nelson, 1974, p 327). These gap junctions reestablish communication. The keratinocytes stop migrating and begin reconstituting the basement membrane (including hemidesmosomes) and the desmosomes and tight junctions between the keratinocytes. Once the basal layer is reestablished, the basal keratinocytes begin dividing, and the upward migration of cells occurs until the appropriate skin thickness is attained (Rovee and Maibach, 2004, p 64). Figures 2–26 through 2–30 illustrate the basal keratinocytes reforming the layers of the epidermis.

FIGURE 2–25

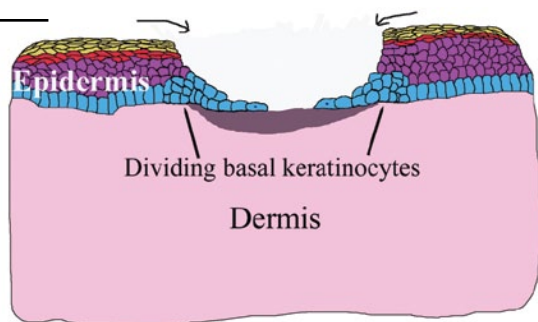*Continued migration of basal keratinocytes and production of new keratinocytes.*

FIGURE 2–26

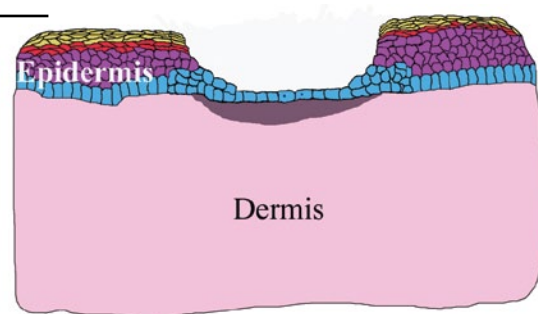*Migrating basal keratinocytes meet in the middle of the wound and reconstitute the basement membrane.*
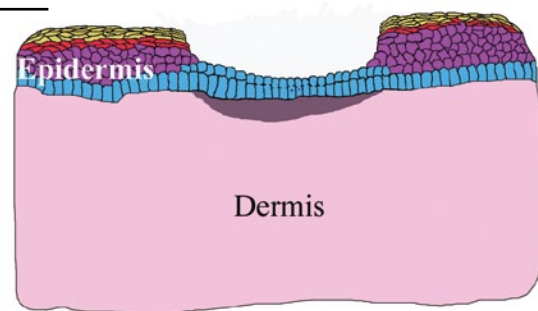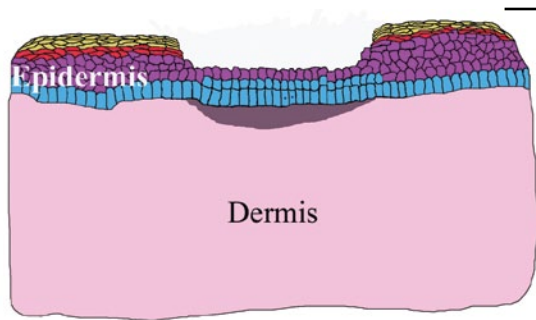
FIGURE 2–27

*Newly formed basal layer begins dividing to reconstitute the upper layers.*

Once the appropriate barrier has been formed, the scab formed by the blood clot during Phase I is released, and the skin returns to its normal physiological state. The friction ridges are not reconstituted. The new basal layer of keratinocytes covering the wound forms the new template for the epidermis at that site. No primary or secondary ridges are formed; consequently, the epidermis does not regenerate the surface ridges and furrows. Additionally, sweat glands are not re-formed. When the sweat glands are damaged as a result of the injury, the cells of the gland also migrate to cover the wound, and the glands are lost (Freinkel and Woodley, 2001, p 284).

2.4.2.3 Phase III: Tissue Remodeling. Once the epidermis has resurfaced, Phase III begins in the dermis. The dermis continues to remodel and reinforce the scar tissue for weeks or months after the injury (Freinkel and Woodley, 2001, p 292).

2.4.2.4 Friction Ridge Skin Wound Healing Model. Figures 2–31 through 2–40 are diagrams created to illustrate wound healing in friction ridge skin. The skin undergoes the same series of events described above, but this model will focus on the layers, rather than the cells, as the skin heals. The layers of the epidermis are color-coded the same as Figure 2–4 (stratum corneum—yellow, stratum lucidum

FIGURE 2–28

Keratinocytes undergo differentiation as they are pushed toward the surface.
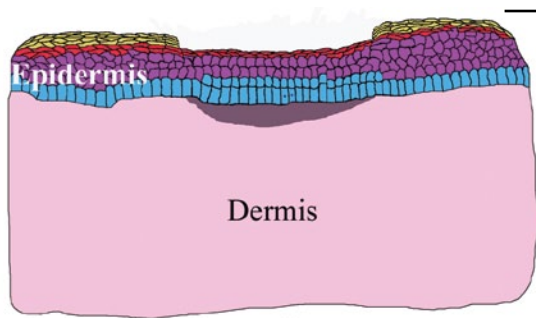
FIGURE 2–29

Continued differentiation of the keratinocytes.
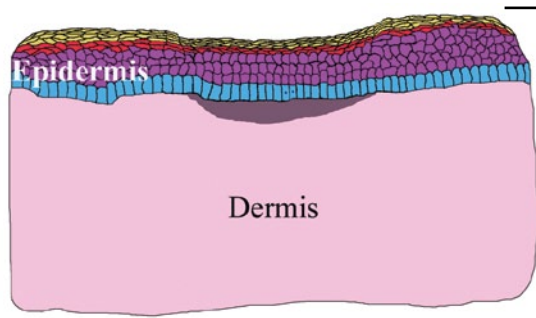
FIGURE 2–30

New epidermis is completely formed.

—orange, stratum granulosum—red, stratum spinosum—dark pink, stratum basale—blue, dermis—light pink), and the friction ridge skin is viewed from three-dimensional and aerial perspectives.

2.4.2.5 The Outer Surface of Scars and the Resultant Impressions. The formation of the scars explains what is seen on the skin and subsequently on the impressions left by the skin. Scars may appear as a void, or may contain partial voids, in an impression because all or part of the newly formed epidermis sits below the level of the surface ridges. Like friction ridges, scars are three-dimensional structures with surface contours and edges. Also like the

friction ridges, the features of the scars will have some variability in appearance, depending on deposition pressure and movement. Figure 2–41 (p 2–24) is an image of a finger bearing a mature scar and an inked impression of the same finger.

2.4.2.6 Uniqueness of Scars. Scars are unique for the very same reason the friction skin is unique: developmental noise (i.e., chance events that occur during development). Richard Lewontin, research professor at Harvard University, describes developmental noise in the following manner: "Wherever cell growth and division are involved, we can expect such noise to contribute its effects. The

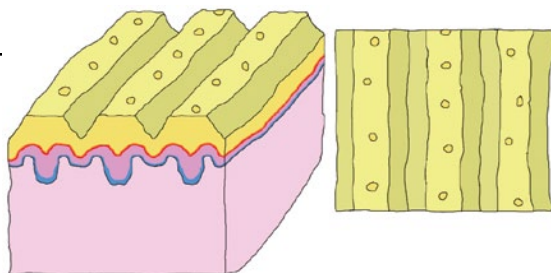**FIGURE 2–31**

Intact friction ridge skin.
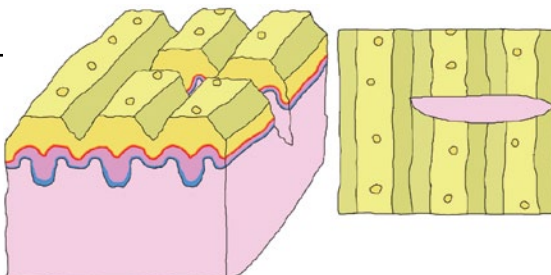
FIGURE 2–32
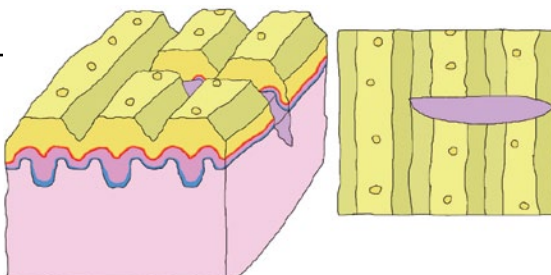
Injured friction ridge skin.

FIGURE 2–33

Repair of the dermis.

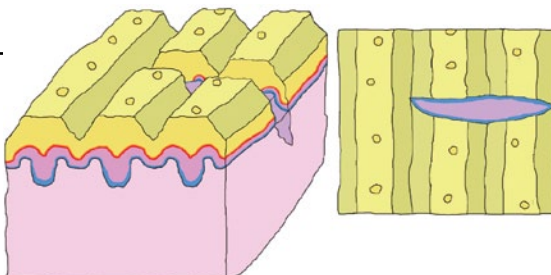**FIGURE 2–34**
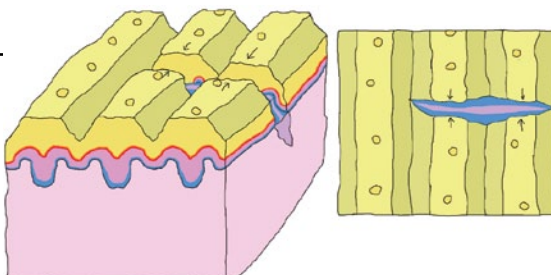
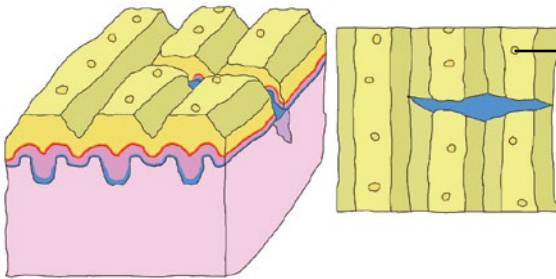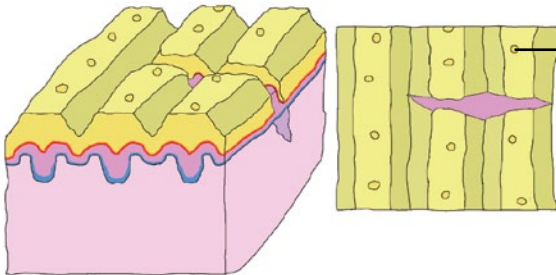*Initial migration of basal keratinocytes.*

FIGURE 2–35

Continued migration of basal keratinocytes and pinching of upper layers of skin.
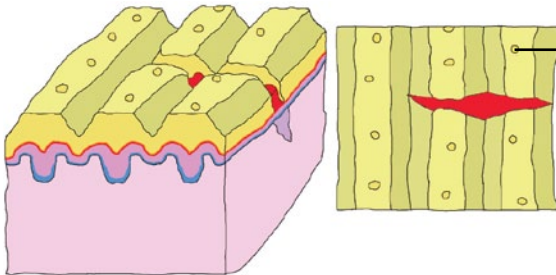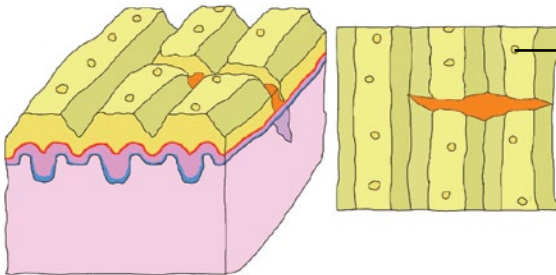
FIGURE 2–36

Final migration of basal keratinocytes and reconstitution of the basement membrane.
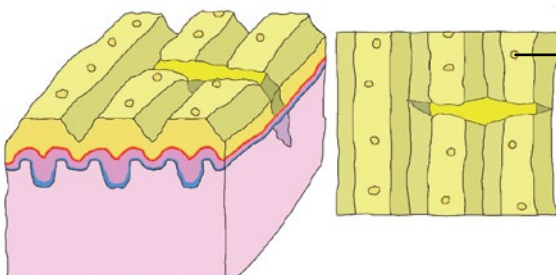
FIGURE 2–37

Basal keratinocytes begin dividing and new cells differentiate to form the stratum spinosum.

FIGURE 2–38

Basal keratinocytes continue to divide and the cells continue to differentiate, forming the stratum granulosum.

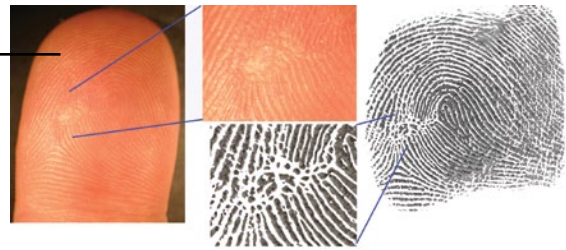## FIGURE 2–39

Formation of the stratum lucidum.

FIGURE 2–40

Complete repair of the epidermis, forming a nonridged scar.

FIGURE 2–41

Mature scar on the friction ridge skin and the resulting impression with ink (position reversed).

FIGURE 2–42

Known prints of a subject taken in July 1990 and September 2004.

exact placement of hair follicles on our heads, the distribution of small moles on our bodies, a hundred such small details of our morphology, are largely under the influence of such random events in development" (Lewontin, 1995, p 26).

When the friction ridges are forming on the fetus and when the basal keratinocytes are activated by an injury, they are under the influence of developmental noise. The cells are rapidly proliferating and are tasked with forming the fetal skin or reconstituting injured skin. These cells are guided but not given specific instructions on their position in the epidermis. In the case of an injury, the cells rapidly proliferate and migrate. The reconstitution of the stratum basale (the new template for the surface) and the effects on the surrounding epidermis (pinching) are the result of this guided, yet random, process. Two injuries cannot duplicate the same scar (Maceo, 2005, p 160).

2.4.2.7 Persistence of Scars. Scars persist for the same reason that the friction ridges persist: attachment sites and regulation of keratinocyte mitosis. The basal keratinocytes regenerate the basement membrane, reestablishing the attachment of the epidermis to the dermis. The keratinocytes also reestablish the cell-to-cell attachments: desmosomes and tight junctions. The keratinocytes resume communication with each other; with the melanocytes, Langerhans, and Merkel cells; and with the dermis.

Communication allows for homeostatic regulation of cell division in the basal layer, ensuring that the epidermis retains its appropriate thickness. As the cells divide, they

move outward in concert and maintain the surface features of the scar (Maceo, 2005, p 160). The impressions in Figure 2–42 were taken more than 14 years apart and demonstrate the persistent nature of scars.

2.4.2.8 Comparison of Impressions Bearing Scars. The use of scars in the comparison of friction ridge impressions has the same basis, and follows the same application, as the use of friction ridges. Once formed, scars are unique and persistent. When an impression of the skin is made, the features of the scar will be reproduced at varying levels of clarity. The clarity of the detail in the impression may reveal the overall configuration of the scar, the position (path) of the scar in the skin, and detailed edge shapes of the scar. This detail makes the scar itself useful in the examination of friction ridge impressions.

2.5 Conclusion

The persistence of the friction ridge skin is explained by the physical attachments of the skin and by the regulated replacement of cells lost at the surface of the skin. The persistent nature of the friction ridge skin makes it an ideal anthropological feature to use as a means of identifying individuals. The structure of the skin also provides a mechanism through which to describe distortion. Variation in the appearance of friction ridge impressions is due to the flexibility of the skin. Understanding that the skin distributes pressure into the more flexible furrows offers valuable insight during the analysis of friction ridge impressions.

Despite its durability, the friction ridge skin is subject to injury and aging. Understanding the aging process provides a basis for variation in appearance of impressions from the same source taken many years apart. Aging processes are particularly critical when explaining the loss of the minute details along the edges of the ridges and the existence of wrinkles. The response of the skin to an injury and the later maintenance of the newly formed skin (scar) provide a basis for the unique features and persistence of scars. The unique and persistent nature of scars allows for their use during the examination of friction ridge impressions. The manner in which skin injuries heal provides an explanation for the variation in appearance of impressions of the skin before and after the injury.

To rely upon the friction ridge skin as a means to identify people, it is necessary to understand why the impressions of the friction ridge skin can be used and what the physical limitations of the friction ridge skin are. If the variation in appearance between two impressions of the friction ridge skin goes beyond the physical limitations of the skin, the impressions cannot be from the same source.

2.6 Reviewers

The reviewers critiquing this chapter were Jeffrey G. Barnes, Patti Blume, Mary Ann Brandon, Brent T. Cutro, Sr., Lynne D. Herold, Andre A. Moenssens, Michelle L. Snyder, John R. Vanderkolk, and Kasey Wertheim.

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